REMARKS

Claims 20 to 24 are pending in the present application. The following remarks and supporting documents are presented in support of the patentability of the pending claims.

Rejection under 35 U.S.C. § 102(b)

In the Office Action, claims 20-22 were rejected under 35 U.S.C. § 102(b) as being anticipated by Nakagawa et al (Cancer Research, 1988, Vol.48, pp.2096-2100) as evidenced by the abstract of Zabel et al (Histology and Histopathology, 1997, Vol.12, pp.283-289). Applicant respectfully traverse the rejection and submits that claims 20-22 are patentable within the meaning of 35 U.S.C. § 102(b). Reconsideration and withdrawal of the rejection are respectfully solicited.

The present invention is directed to methods for inducing the re-expression of a previously silenced gene encoding the human sodium/iodide symporter in a human thyroid carcinoma cell by administering a compound selected from 5-azacytidine, sodium butyrate, dimethylsulfoxide, S-adenosyl-1,8-diamino-3-thio-octane, and phenylacetate. Applicant's studies have shown that these demethylating agents or differentiating agents can restore transcription of a previously inhibited iodide transporter gene, thus restoring iodide transport. Moreover, applicant's studies have demonstrated restoration of gene expression in a variety of human thyroid carcinoma cells, for example in follicular carcinoma cells. Therefore, the claimed invention provided a method for treating thyroid cancer cells which have lost the ability to transport iodine and which have an inactive thyroid specific response element. Applicants have shown that administration of a demethylating agent or differentiating agent to the cells results in expression of the therapeutic response element and uptake of iodine.

In contrast, Nakagawa et al describes that the butyrate treatment induces cultured human medullary thyroid carcinoma cells to acquire *in vitro* properties consistent with the differentiated phenotype of the mature thyroid cell. As mentioned by the Examiner, the TT cell is a parafollicular cell line. Medullary thyroid carcinoma derives from these parafollicular cells, notable for secreting calcitonin and other substances, and constitutes 5% to 10% of thyroid malignancies (see attached documents). However, parafollicular, or C, cells, lie between the follicles of the thyroid gland. These cells do not have the same embryological origin as do the thyroid follicular cells. A parafollicular cell does not need an active gene for sodium/iodide symporters since in this cells there is no active transport of iodide, since in this cells there is no synthesis of thyroid hormones. Therefore, medullary thyroid cancer is untreatable by the uptake of iodine. There is no inducable re-expression of a previously silenced gene encoding the human sodium/iodide symporter. Therefore, the human thyroid carcinoma cells according to the invention differ from the cells used in the cited documents. They are different cell types.

Accordingly, it is submitted that Nakagawa et al does not negate the patentability of the presently claimed invention. Reconsideration and withdrawal of the rejection under 35 U.S.C. § 102(b) are respectfully requested.

Respectfully submitted,

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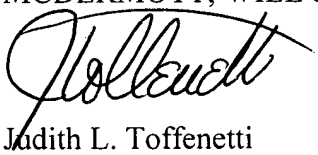

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Date: October 7, 2004